[In the light of our present knowledge of the clinical differentiation between measles and rubella, this conclusion, based solely on a similarity of blood reactions in the two diseases, illustrates the danger of too much hæmatology.—Ed.].

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Experimental Fat Necrosis.—Wells (*Journal of Medical Research*, 1903, vol. iv. page 70) has found that fat necrosis can be produced with constancy, especially in cats and dogs, by intraperitoneal injections of extracts of hog's pancreas or of ordinary commercial pancreatin. Such extracts, moreover, are active either in weak acids, alkalies, or in water. Preparations of carica papaya, or of simple alkaline solutions, did not produce fat necrosis. When the pancreatin was heated for five minutes at temperatures between 65° and 70° C. the power to produce fat necrosis was much reduced, and above 71° C. it was entirely destroyed. These observations pointed to enzyme action as a source of the condition. The exact pancreatic enzyme, which was the cause of the fat necrosis, could not itself be isolated. It was found, however, that trypsin, weak in or devoid of lipase, did not produce this condition, and that, likewise, lipolytic extracts of hog's liver and cat's serum were entirely inactive. Moreover, negative results were obtained with a mixture of pancreatic trypsin and lipolytic extracts of liver. It was not possible to obtain pure pancreatic lipase, so that the action of this enzyme alone could not be studied. Extracts of fresh dog's pancreas, however, which possessed lipolytic power, but which were almost devoid of tryptic power when injected, caused definite fat necrosis; but if to such extracts an emulsion of duodenal mucosa were added, by which means the tryptic activity is greatly increased, no fat necrosis occurred. Thus it appeared that as the pancreatic extracts lost their lipolytic action, their power to produce fat necrosis also diminished; and the author believes it highly probable that the lipase is in some way highly essential to this particular property of the pancreatic extracts. Besides the *intravitam* experiments, many of the features of fat necrosis could be produced with pancreatin in animals after death, and also *in vitro*. The dissemination of the fat necrosis outside the abdominal cavity was sometimes observed as early as twelve hours after intraperitoneal injections, and it was thought that through the lymphatics this spreading was accomplished. From a study of the progressive changes which took place during the production of fat necrosis it was found that the first alteration consisted in death of the fat cells, while not until later did the fat-splitting occur and the formation of crystalline products appear. The author concludes, therefore, that the fat-splitting is subsequent to and not the cause of the necrosis. The fat necrosis proceeded but for a very few hours at any one point, for the extension seemed to be limited by a zone of leucocytes, the ultimate function of which appeared to be the absorption of the products formed.

Finally, the mere presence of fat necrosis did not seem to be detrimental to the lives of the animals, and healing was possible by a proliferation of the connective tissue from the margins of the areas.—W. T. L.

A Contribution to the Pathology of Balantidium (Paramæcium) Coli.—KLIMENKO (*Beiträge zur Path. Anat. u zur Allg. Path.*, 1903, Bd. xxxiii. p. 281) discusses this subject and describes a case of fatal infection by the balantidium coli. The patient suffered with severe diarrhœa, followed by anæmia. Blood and mucus were constantly present in the stools. Parasites, frequently enclosing red blood cells, were, on several occasions, found in the freshly-evacuated feces. At autopsy the mucous membrane of the colon proved to be greatly swollen and congested, while a large amount of mucus covered it. Several ulcers, sometimes reaching to the muscular coat, were seen in the large intestine. Microscopically there was an acute inflammation of the mucous and submucous coats, with small areas of necrosis and hemorrhage in these situations. The infusoria were found, in great abundance, distributed through practically all of the coats of the intestinal wall, but were most numerous in the mucosa and submucosa. The author concludes that the balantidium coli must be regarded as the cause of certain diarrhœas. At first the organisms by their motion produce a diarrhœa from a mechanical irritation of the mucosa. Whether they are still motile after penetrating the wall of the colon is doubtful.

Finally, they give rise anatomically to a catarrhal or ulcerative colitis, but it is as yet uncertain whether the parasites alone are answerable for all the intestinal changes, or whether the schizomyces, also, have some part in the process. It is very probable that the balantidium reproduces in the intestinal wall. Since the infusoria may gain entrance into the bloodvessels, it is not improbable that they form emboli in the various organs, and especially in the liver and lungs. It cannot as yet be determined whether they produce the anatomical lesions principally through their mechanical action or through a chemical irritation.—W. T. L.

Further Observations on the Reaction of Bacillus Pestis in Plague.—Row (*British Medical Journal*, 1903, No. 2210, p. 1076) has investigated the factors concerned in the bacteriolytic properties of blood serum from convalescent plague patients. The serum when first removed is highly bacteriolytic for B. pestis, but either after heating at 56° C. for a few minutes, or after standing at room temperature for several days, the serum becomes entirely inactive. Fresh rabbit's serum, which in itself is not bacteriolytic for B. pestis, was added to the heated serum from convalescent plague